Dissertation on

ROLE OF BOEY`S SCORE IN PREDICTING MORTALITY AND MORBIDITY IN PERFORATED PEPTIC ULCER PATIENTS

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CERTIFICATE

This is to certify that this dissertation on **ROLE OF BOEY SCORE IN PREDICTING MORTALITY AND MORBIDITY IN PERFORATED PEPTIC ULCER PATIENTS** presented herein by **Dr.DARWIN BRITTO .D**, is the original work done in the Department of General Surgery, Government Stanley Medical College and Hospitals, Chennai in partial fulfilment of requirements of M.S. Branch-I (General Surgery) examination of The TamilnaduDR.M.G.R.Medical University to be held in April 2014 under guidance and supervision, during the academic period of 2011-2014.

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DECLARATION

I, **DR.DARWIN BRITTO.D,** solemnly declare that this dissertation, titled **"ROLE OF BOEY`S SCORE IN PREDICTING MORTALITY AND MORBIDITY IN PERFORATED PEPTIC ULCER PATIENTS"** is a bonafide record of work done by me in the Department of General Surgery, Government Stanley Medical College and Hospitals, Chennai under the guidance of my unit chief **PROF.DR. BALAMURUGAN M.S.,** Addl. Prof. of surgery.

This dissertation is submitted to The Tamilnadu DR.M.G.R. Medical University, Chennai in partial fulfilment of regulations for the award of M.S. (General Surgery) examination, to be held in APRIL 2014.

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CONTENTS

ABSTRACT:

The following is a study on role of Boey`s score on predicting the mortality and morbidity in perforated peptic ulcer patients. This study was done in department of general surgery government Stanley hospital. A total of 50 patients admitted with perforated peptic ulcer disease were included in this study after getting their informed consent. For each of these patients Boey score was calculated based on following three criteria

Time since perforation leesses or greater than 24 hours

Presence or absence of shock

Associated medical illness

Based on these a score of 0 to 3 was assigned to each patients and the patients were followed up post-operatively for development of morbidity and mortality. The results were then analysed statistically.

KEYWORDS:

Perforated peptic ulcer

Boey score

INTRODUCTION

Perforation of a peptic ulcer is considered as one of the major complication of peptic ulcer disease. This is due to the fact that perforated peptic ulcer is a surgical emergency and leads to increased morbidity and mortality. The risks associated with a perforated peptic ulcer can be predicted with a number of scoring systems. One such scoring system which is simple and used in predicting mortality and morbidity in a perforated peptic ulcer is Boey scoring system.

This study is for validation of Boey score in predicting mortality and morbidity in our setup.

AIMS AND OBJECTIVES

The purpose of the present study is topredict morbidity and mortality in patients with perforated peptic ulcer using Boey score.

REVIEW OF LITERATURE

HISTORY OF PEPTIC ULCER PERFORATION

For many years, people who were healthy suddenly developed abdominal pain, abdominal distension, vomiting and died within a period of few hours to days. This was the time when perforation of peptic ulcer was not heard of and the symptoms were attributed to poisoning and there were occasions where people have been sent to prison.

The first documentation of perforated peptic ulcer happened about 2000 years ago. The body of a man who died in the year 167 BC who belonged to west Han dynasty was preserved by keeping the body in Chinese herbal solution. In the year 1975, archeologist from china

discovered this coffin in Hubei province which remained well preserved. This remains were studied in pathology department of Wu Han medical college by Wu Zhongbi. The cause of death was found to be peritonitis that resulted from prepyloric ulcer perforation.

In the year 1670, daughter of king Charles I ,Henrietteanne died suddenly and unexpectedly at a age of 26, after a day of suffering from abdominal pain and tenderness. Suspecting poisoning autopsy was performed on her. Autopsy revealed peritonitis and also showed a small hole in the anterion portion of stomach. But doctors of King Charles had never heard of perforated peptic ulcer and they thought that the hole in stomach was accidently caused by knife of the dissector.

Since the time autopsies were allowed, many a time perforation of stomach was observed. It was in the year 1817 that Benjamin Travers described acute peptic ulcer perforation in a group of patients.

In the year 1843, Edward Crisp was the first to describe syptoms of perforated peptic ulcer after evaluating 50 cases

It was in the year 1884, John Mikulicz-Radecki, a student of Theodore Billroth, performed closure of perforated peptic ulcer. Inspite of his efforts the surgery was unsuccessful as the patient died.

The first successful surgery was performed in the year 1892, may 19th , by Ludwig Heusner in Barman, Germany. Heusner closed the perforation in a time of 2.4 hours.

EMBRYOLOGY OF STOMACH AND DUODENUM

During the 4th week of embryo, the stomach starts to form as fusiform dilatation of the foregut. This developing stomach is attached to body wall by means of ventral and dorsal mesenteries.

By 5th week, the dorsal wall of stomach tends to grow faster when compared to that of ventral wall. This differential growth results in the formation of lesser and greater curvatures of stomach.

During 7th week of intrauterine life, stomach rotates 90 degress in a clockwise manner about a longitudinal axis. The lesser sac or omental bursa which is a space behind the stomach is produced as a result of this rotation. The lesser curvature now faces right and greater curvature faces left.

By 8th week, the stomach along with duodenum rotates in aantero-posterior or ventrodorsal axis, which leads to pulling the end of stomach upwards. This rotation pulls up the duodenum into a c-shaped position.

The dorsal mesentry becomes thinned out and hangs from greater curvature of stomach which is called greater omentum. The ventral mesentry gets attached to developing liver and is called lesser omentum.

HISTOLOGY OF STOMACH AND DUODENUM

The layers of stomach are as follows

The mucosa:

This is the innermost layer, which is thrown into numerous folds called rugae. On mucosal surface there are small, funnel-like depressions called gastric pits. Gastric glands open into these pits.

The epithelium on the surface of mucosa is simple, tall columnar. This consists of surface mucous cells which produces an alkaline mucus which protects the underlying epithelium.

Cardiac glands:

Heavily branched tubular glands are present here, which produce mucous. Only few secretory principal galnds are found here.

Corpus-fundic or principal gland:

Each of these glandular tubule consists of body, neck and isthumus. There are four types of cells found here. They are

Chief cells:

They are also called zymogenic cells, they are the most abundant of the four types of cells found . The role of the chief cells is to secrete pepsinogen.

Parietal cells:

They are otherwise called oxyntic cells. They occur in the neck of glands. Their main function is to secrete hydrochloric acid and intrinsic factor which is important for vitamin B12 absorption.

Mucous neck cells:

As their name implies they are located in the neck and also secrete mucous.

Endocrine cells:

These cells form a part of GEP (gastro-entero-pancreatic) system. The endocrine cells present in the stomach are G and D-cells, which secrete gastrin and somatostatin respectively.

Pyloric glands:

The glands in pyloric region are coiled as compared to principal glands and also they are branched. A few parietal and gastrin producing G-cells are found here.

Lamina propria:

This is formed by loose connective tissue. It consists of lymphocytes, fibroblast, eosinophil, macrophages.

Muscularis mucosa:

This consists of both circular and longitudinal muscle cells. The circular being the inner layer and longitudinal the outer layer.

Submucosa:

This layer is situated deep to the mucosa. It consists of loose connective tissue.

Large blood vessels, nerves and the lymphatics are located in this layer.

Muscularispropria:

This is also called muscularisexterna. This consist of three layers of muscle cells the innermost is the oblique layer, middle layer is made of circular muscle and the outermost layer is made of longitudinal muscle.

Duodenum:

The mucosa:

The mucosa forms numerous intestinal villi. These villi are lined by columnar type of epithelium. The epithelial cells inturn form numerous microvilli. Inbetween these intestinal villi we can see openings of tubular glands which are called crypts of lieberkuhn. The function of these is to secrete intestinal juice which contain the enzyme enteropeptidase. The other cells present here are gastrin and somatostatin producing cells and also cells which secrete cholecystokinin.

Lamina propria and muscularis mucosa:

This is similar to that found in stomach.

Submucosa:

Duodenum contains specialized glands called brunner`s gland. The main function of these glands is to secrete mucous and this mucous is bicarbonate- rich to maintain a pH about 7-8. This mucous protects the duodenum from effects of gastric acid.

Muscularispropria:

This is similar to that as found in the stomach.

ANATOMY

The stomach is like a pouch that connects the abdominal oesophagus and also the first part of the small Intestine, duodenum.From a surgical point of view, however, we are going to embody the gastroesophageal junction and also the initial portion of the duodenumwith the stomach. These are the proximal and distal internal organconnections that form a part of what is known as the "surgical stomach".

Topography:

The stomach obliquely lies in the left and upper part of abdomen occupying the epigatrium, left hypochondriac and umblical regions.

Few abdominal organs are as mobile as stomach. Its positiondepends on the position of the individual, the degree to that thestomach is distented, degree to that the bowel is distented, the tone ofthe wall, and also the habitus of the patient. The distended stomach ishigher in broad, heavyset people than that in slender people.

The only fixed point of reference for stomach is the musculargastro-esophageal junction, which lies to the left of the midplanebehind seventh costal cartilage at the level of tenth dorsalvertebra.

External features:

It has 2 orifices, 2 curvatures, and 2 surfaces.

The cardiac opening is joined by lower part of oesophagus.

The pyloric orifice opens into duodenum.

The lesser curvature that forms the right border of stomach is concave. Incisuraangularis is the most dependent part of lesser curvature The greater curvature which forms the left border of stomach is convex.

The anterior surface of the stomach faces in an upward and also forward direction.

The posterior surface of the stomach faces in a downward and backward direction

Subdivisions:

The stomach is subdivided into 2 parts i.e the cardiac and pyloric

parts by a line drawn from the incisuraangularis downward and to the left. The cardiac part is again subdivided as fundus and body, thepyloric part subdivided as pyloric antrum and pyloric canal.

The fundus is a dome shaped region located above a horizontal line drawn from cardiac orifice level.

The body lies between fundus above and antrumbelow . It candistend along the greater curvature enormously.

The distinction between the pyloric antrum and pyloric canal is by sulcus intermedius. This is an inconstant sulcus present along the greater curvature.

The pyloric canal has a length of about one inch. It is narrow and also tubular.

Relations of stomach :

Anterior relation:

The stomach is in relation with the liver, diaphragm, and anterior body wall.

Posterior relation:

The stomach lies in a bed shaped by the diaphragm, spleen, left kidney suprarenal gland, pancreas, transverse mesentery, and lienal flexure of the colon.

Peritoneal relation:

The peritoneum lines the stomach on both its surfaces. Theperitoneum lining the anterior and posterior surface unite at the lesser curvature end to become continuous with the lesser omentum. Similarly at the greater curvature end the peritoneum lining stomach becomes continuous with the greater omentum.

At the greater curvature near its cardiac end the two layers meet to form gastro -splenic ligament. The gastro-phrenic ligament is formed by reflection of peritoneum on posterior surface of stomach on to thediaphragm. Above the gastro-phrenic ligament a small portion stomach lies directly on the left crus of the diaphragm which is called the `Bare area of the stomach .

Blood supply of stomach:

The stomach`s blood supply is from the following arteries

1. Left gastric artery [coeliac trunk branch]
2. Right gastric artery [common hepatic arterial branch]
3. Right gastroepiploic artery [gastroduodenal arterial branch]
4. Left gastroepiploic artery [splenic arterial branch]
5. five to seven short gastric arteries [splenic arterial branch]

The right and the left gastric arteries run along the lesser curvature supplying it. The right and the left gastroepiploic arteries lie along the greater curvature supplying it.

Lymphatic drainage of stomach:

The stomach`s lymphatic drainage is by means of following four lymph node groups

1. Left gastric nodal group, this follows the left gastric artery. They ultimately drain into the celiac group of nodes. They drain the lesser curvature portion of stomach .
2. Leftgastroepiploic and short gastric vessel nodal group. The lymphatic vessels of left side of the greater curvature region of stomach follow these vessels and drain into them . From these nodes further drainage is into the pancreatico-splenic group of nodes.
3. Rightgastroepiploic nodes, drain right half of the greater curvature region of stomach upto the pylorus.
4. Pyloric nodes drains the pyloric region of stomach. From heredrainage of these nodes is into the hepatic and left gastric nodes

Nerve supply of stomach:

The stomach`s nerve supply is by both sympathetic and the parasypmpathetic system

Sypmpathetic system:

The sympathetic supply is derived from T6 to T10 segments ofspinal cord through greater splanchnic nerves , celiac and through the hepatic plexus.

These nerves are

1.vasomotor

2.motor supply to pyloric sphincter and a inhibitory effect to remaining portion of gastic musculature

3.main pathway of pain sensation from stomach

Parasympathetic system:

The vagi are the parasympathetic supply. The anterior and posteriorvagi supply the stomach which are the left and right vagirespectively.

The anterior vagus gives off a number of gastric branches andtwo pyloric branches.

The posterior vagus gives of gastric branches and celiac branchesThey function as

1.secrotomotor for secretion of gastric juice which is rich in pepsin and hydrochloric acid

2.motor to gastic musculature and inhibitory to pyloric sphincter.

GASTRIC PHYSIOLOGY

The stomach functions to prepare the food that is being taken for the purpose of digestion and absorption before the food enters duodenum. During the initial stages when the food is being digested , the solid components present in the meal gets stored for many hours while they are reduced in size and brokendown into smaller components.

The proximal part of stomach is responsible for its storage function .This by means of a process called receptive relaxation. This is a process that cause passage of liquids easily along lesser curvature, meanwhile solid food particles settledown along greater curvature .

Emptying of solid food is aided by antrum, which functions to act as a pump so that solid components present in the food pass via the pylorus. The antralpump along with the pylorus function in a well co-ordinated way, so that passage of food component are appropriate for delivery into the duodenum.

The stomach also participate in digestion of meal. Peptic digestion also aids in metabolizationofdifferent components present in the food that is being taken in.

Gastric Function – its regulation:

Gastric function gets regulated by means of neural and hormonal factors . Hormones that mediate gastric function are amines or peptides which upon interaction with respective target cells mediate their effects by any of the following ways: endocrine, neurocrine, or paracrine. The resultant effect of this target cell is dependent upon the balance between endocrine, neurocrine, and paracrine signals converging on the cells.

Peptides

1.Gastrin

Gastrin is secreted by G cells which are located in antrum of stomach . Many molecular forms of gastrin exist, but around 92% of gastrin released from antrum is a peptide with 17–amino acids. The gastrin release is stimulated by a meal. Presence of luminal acid inhibits gastrin release. Somatostatin which has a paracrine action on G cells present in antrum causes inhibition of release of gastrin .

Gastrin regulates the gastric phase of secretion of acid that follows food intake. The parietal cells have receptors for gastrin but it has been found that histamine that is secreted by enterochromaffin-like (ECL) cells ,on gastrin stimulation , is responsible for mediation of acid secretion. Thus gastrin causes secretion of acid indirectly through histamine release.

2.Somatostatin

Somatostatin , produced from D cells , exist endogenously either as 15– or 29–amino acid peptide. Molecular form that occurs predominantly in the stomach is somatostatin-15. Somatostatin is secreted by cells present in fundus and antrum.

Somatostatin directly inhibit acid secretion by parietal cells . It indirectly causes inhibiton of secretion of acid by inhibiting release of gastrin and decreasing release of histamine from ECL cells.

The main stimulus causing somatostatin release is presence of acid . Acetylcholine produced from stimulation of vagal fibers inhibit somatostatin release.

Effects of *H.pylori* on Somatostatin

H. pylori causes a reduction in population of antral D cell, which inturn leads to decrease in levels of somatostatin . This decrease in levels of somatostatin lead to G cells disinhibition with increase in release of gastrin and hence an increased acid production. *H. pylori* eradication helps in restoration of population of D-cell, resulting in increased somatostatin release .

Histamine

Histamine plays an important role in stimulation of parietal cells . H_2-receptor blocker administration almost causes complete abolishment of secretion of acid in response to stimulation, by either gastrin or acetylcholine. This suggests that histamine is necessary as aintermeditory of acetylcholine and gastrin stimulated secretion of gastric acid.

Histamine is found in ECL (enterochromaffin-like) cells and also in mast cells resident in stomach. The release of histamine occurs upon stimulation by acetylcholine and gastrin. Somatostatin inhibit release of histamine by indirectly inhibiting action of gastrin by interaction with receptors present on enterochromaffin cell.

Gastric Acid Secretion

The three stimuli regulating secretion of gastric acid by parietal cell are:

Gastrin

Acetylcholine

Histamine

These three stimuli are responsible for both basal and stimulated secretion of gastric acid. Acetylcholine , a neurotransmitter released from vagal nerve endings is responsible for secretion of acid . The fibres from Vagusinnervate the parietal cells, the G cells and also the ECL cells to mediate release of peptides from each of these cells.

Basal Acid Secretion

Even when food is not present in stomach, that is when the stomach is empty, there is always some amount of basal secretion of acid which accounts to about 10 to 14% of maximal output of acid. Basal secretion of acid exhibit a circadian variation, in which acid secretion during night is greater compared to day. In basal conditions, there is secretion of about 1 to 5 mmol/hour of hydrochloric acid.

Thebasal acid secretion is because of combined input from histaminergic and cholinergic stimuli.

Stimulated Acid Secretion :

Food intake is a physiological stimuli for secretion of acid .The three phases of acid secretion following food intake are :

Cephalic phase

Gastric phase

Intestinal phase

Cephalic Phase

This phase starts with the sight, thought, taste, or smell of food, that inturn causes stimulation of neural centers located in cortex and hypothalamus. These are the centers responsible for transmission of signals to stomach through the vagal nerves, that releases acetylcholine . Acetylcholine increases acid secretion by parietal cell stimulation . The cephalic phase account for about 25% to 32% of total acid production following a meal .

Gastric Phase

The gastric phase starts with food entering the lumen of stomach. The food which is ingested interact with antral G cells to cause release of gastrin . Mechanical distention of stomach by ingested food also stimulates acid secretion by activating receptors like stretch receptor found in the stomach to stimulate vago-vagal reflex . This arc can be abolished by proximal gastric vagotomy .

Distention of antrum by food also cause release of gastrin , this is known as *pyloro-oxyntic reflex.* The gastric phase account for 60%-70% of acid output since it lasts for a longer time till the stomach becomes emptied devoid of food.

Intestinal Phase

This phase of secretion of acid begins following entry of chyme into duodenum. This accounts for 10% of the acid secretion. It has been postulated that there is a unique hormone called entero-oxyntin that is released from mucosa of small bowel . This hormone may be responsible for intestinal phase of secretion of acid.

Gastric Acid – Functions:

1. The acid secreted by stomach play an important role for digestion of a meal. It is needed for convertion of pepsinogen into pepsin. This pepsin inturn causes hydrolysis of proteins to polypeptides.

2. Gastric acid is responsible for secretin release from the duodenum, this secretin causes secretion of bicarbonate rich pancreatic juice.

3. Gastric acid plays a important role in limiting the colonization of upper GI tract with bacteria. Alkalinizationof gastric lumen decreases the bactericidal effects of acid. This inturn creates a milieu which is favourable for overgrowth of bacteria.

Gastric Juice

Gastric juice, whose secretion volume is about 1.1 to 1.5 litres/day is produced as a result of secretion by chief cells, parietal cells, and mucus cells in addition to this it also contains swallowed saliva and duodenal refluxate.

Parietal cells are responsible for secretion of a solution that is isotonically similar to plasma, containing 165 mmol/L. This solution

has a pH of 0.9. However, the lowest pH found in stomach is about 1.6-1.9. This is because of dilution of secretion of parietal cells by means of gastric secretions by other cells, which contain potassium, sodium ,bicarbonate etc.

Intrinsic Factor

This is a 65,000-dalton protein that is secreted by the parietal cell. Intrinsic factor is primarily required for vitamin B_{12} absorption in ileum.

Pepsinogen

Pepsinogen is a proteolyticproenzyme having a molecular weight of 42,500 . Pepsinogen is secreted by chief cells and also in small quantities by mucus neck cells . Acid presence is required for conversion of pepsinogen to pepsin by means of removal of a amino-terminal peptide. This pepsin is a proteolytic enzyme which becomes inactivate if the pH becomes greater than 5.

Mucous and Bicarbonate

Together, mucous and bicarbonate combine, and causes neutralization of gastric acid at the gastric mucosa. Their secretion is by means of surface mucous cells and mucous neck cells . Mucous is like a viscous and elastic gel which has about 90% water and 10% glycoproteins.

Mucous acts as a mechanical barrier preventing damage to epithelium. Mucous production can be stimulated by cholinergic agonists, prostaglandins. In contrast to this, NSAIDs (non steroidal anti-inflammatory drugs) and anticholinergic drugs inhibit secretion of protective mucous. *Helicobacter pylori*, secretes numerous proteases which causes breakdown of the mucin layer and interfere with the barrier function of mucous layer.

Bicarbonate secretion occur by both active and passive means. The amount of bicarbonate that gets secreted is less as compared to secretion of acid in the stomach.

PEPTIC ULCER DISEASE

Epidemiology

PUD, still probably remain as one among the most common and costly gastrointestinal tract disease.

The incidence of peptic ulcer including both gastric as well as duodenal ulcer in USA is around 1.9%. In addition to this, there occurs about 4 million ulcer patients with recurrences yearly.

In the recent past, the elective admissions for PUD have decreased to a large extent but the admission for sake of complications resulting from ulcer shows only little change and remains the same. It has been found that more than 135,500 surgeries for PUD are being done annually and that around about 9000 patients die of complications related to PUD yearly.

In India the point prevelance of peptic ulcer disease is estimated to be 4.62% and the lifetime prevelance is about 11.23%.

Helicobacter pylori discovery represent a dramatic change in understanding the pathophysiology of PUD and has made many experts to come to a conclusion that PUD is an infectious disease.

Consequently, any treatment plan devised for PUD, either medical or surgical, requires that *H. pylori* is being taken account.

PATHOGENESIS

Helicobacter pylori Infection

It is estimated that H.pylori infection is associated with 92% of patients with duodenal ulcer and about 70% of gastric ulcer patients . When H.pylori is eradicated by treatment , recurrence of ulcer seems to be an extremely rare occurence.

Helicobacter pylori is spiral or helical in shape. It is a rod shaped gram-negative organism with 3 to 5 flagella. It resides in the epithelium of gastric mucosa just beneath the mucous layer, that gives protection for the bacteria against the effects of acid and antibiotics.

H.pylori`s movement through the mucus layer is aided by its shape and flagella. It also produces a number of enzymes that help the bacteria in its adaptation to the hostile acidic milieu of the stomach.

H.pylori is considered as one among the bacterias which are potent producers of the enzyme urease. Urease splits urea converting it into ammonia and bicarbonate. This creates an environment that is alkaline around itself in the setting of an environment of gastric acid . This organism is microaerophilic in nature. The temperature required for isolation of this organism around is 36°C to 38°C. Time taken for growth of the organism ranges from about 3 days to 6 days.

Helicobacter pylori has a high affinity to gastric epithelium and can only live in gastric epithelium .This is due to the fact that only the epithelium of stomach express certain types of adherence receptors. These receptors are specific and are recognized only by helicobacter pylori . Similarly, heterotopic gastric mucosa found in esophagus ,in places where gastric metaplasia occur in duodenum , Meckel's diverticulum have been found to harbour the organism.

The mechanisms that cause *H. pylori*–mediated GI insult is by three potential means which are as follows:

1. Toxic products produced by the bacteria which causes injury to local tissues.

2. Immune response being locally induced at mucosa

3. Increase in levels of gastrin which causes an increase in secretion of acid

The toxic mediators that are locally produced by h.pylori are breakdown products of urease activity like ammonia, certain cytotoxic products, a mucinase which degrade mucous, a phospholipase which damage the mucous and cell walls, and platelet-activating factor, that causes injury to mucosa and also microcirculatory thrombosis.

H. pylori–mediated immune response at tha mucosal level also lead to GI insult.*H.pylori* cause local inflammation in gastric mucosal region and produces chemotactic factors which will cause attraction of cells like neutrophil and monocytes. These inflammatory cells on

activation, will result in production of numerous cytokines that are proinflammatory and also reactive oxygen metabolites.

Helicobacter pylori infection causes basal and stimulated gastrin levels to increase, probably because of a reduction in number of D cells in antrum . This relation between secretion of acid and *H. pylori* does not occur in a straightforward manner. Healthy volunteers who are positive for H.pylori have a very small or no increased production of acid when compared with that of volunteers who are *H. pylori*–negative

Peptic ulcers have a strong association with antral gastritis. A number of studies which were done in pre-*H.pylori* era showed that nearly all patients with peptic ulcer had evidence of antral gastritis on histological examination. It was also found that the patients who had only gastric ulcers without gastritis were patients taking aspirin.

Now it has been found that most, if not all patients with evidence of histologic gastritis are because of *H. pylori* infection. The infection initially appears to be limited to antrum and causes inflammatory response in antrum.

An evidence that supports the role of *H. pylori* in causing histologic gastritis is from two physicians who voluntarily ingested

H.pylori inoculum after confirmation of normal gross as well as microscopic gastric mucosa before ingestion. This resulted in these two men developing *H. pylori* infection. This inturn resulted in acute inflammation which was observed on fifth and tenth day. On 14^{th} day, it was replaced by chronic inflammatory cells . This report provides documentary evidence that *H. pylori* causes histologic gastritis.

Helicobacter pylori leads to a chronic infection found all over the world. As soon as a person is infected, the infection is likely to remain lifelong since spontaneous remission is a rare occurence. The infection and socio-economic status of the patient appears to be inversely related. The reason being factors like familial clustering, crowding and sanitary conditions. Infection with H.pylori is most common in low socioeconomic group (84%), intermediate in middle socio-economic group (51%), and is low in high socio-economic group (12%).

In developing countries like India high rates of infection with *H. pylori* is found in children. There seems to occur a linear, gradual and steady raise in acquiring of infection with H.pylori with age.

H. pylori infection has a association with numerous common upper GI disorders. Infection with H.pylori appears to be almost always

present in patients with chronic active gastritis . The association of H.pylori infection in patients having non-ulcer dyspepsia ranges to around 60%.

Most patients with gastric cancer patients have evidence of infection by *H. pylori* in the past. Though this association appears to be strong, causal relationship is yet to be proven. H.pylori infection has been implemented in the occurrence of MALToma or mucosa associated lymphoid tissue lymphoma. It is interesting to find that these lymphomas regress after eradication of H.pylori; hence, H. pylori eradication is always tried before starting the patient on chemotherapy.

Data available for estimation of lifetime risk for development of an ulcer following infection by H.pylori is limited. In serological studies done in Austria with an evaluation period of 20 years, in 16% of *H. pylori*–seropositive patients, development of duodenal ulcer was observed, when compared with that of only 4% of seronegative individuals developing an ulcer.

Nonsteroidal Anti-inflammatory Drugs

Next to *H. pylori* infection, NSAIDs intake is considered as the most common cause of PUD. The increased NSAID use occur mostly in women above 50 years of age, this is also the group of patients having an increased incidence in bleeding from peptic ulcers. This increased risk for ulceration and bleeding is directly proportional to daily dosage of NSAID intake. In addition to this, the risk for complications also increases with age above 60, patients with a prior GI event, or simultaneous use of steroids or anticoagulants.

As a result, the NSAIDs intake remain as an important factor in pathogenesis of peptic ulcer , and hence in development of ulcer related complications like bleeding, perforation and death . NSAID`s role in PUD becomes more evident if we consider that about 4 million people in the United states of america take NSAIDs daily, leading to 2 in 10 of these patients , landing up having an acute ulcer. In addition to this, 3% to 5% of patients using NSAIDs develop ulcer related complications annually, and more than 3500 patients die .Around 24,000 hospitalizations annually, are attributable to GI complications from NSAIDs. When compared with general population, the risk in NSAID user is 2- to 10 times for GI complications.

NSAID intake causes both acute and chronic gastroduodenal injury . This risk for ulcer formation is directly related to anti-inflammatory potential attributed to each NSAID.

The acute lesions appear in 1 to 2 weeks of NSAID intake and can range from hyperemia of mucosa to superficially occurring erosions of gastric mucosa.

Chronic injury usually occur after a period of about 4 to 5 weeks . These can be seen either as erosion or ulceration occurring in antrum of stomach or in the duodenum.

When compared to ulcers resulting from H. pylori infection , that occur most often in duodenum, ulcers from NSAID origin commonly occur in the stomach. Stoppage of NSAID usage, prevents ulcer recurrence.

Acid :

There appears to be a linear relationship between parietal cell number and maximal acid output. The secretory rates of gastric acid are different in patients having upper GI diseases. This acid secretory rates of stomach show a marked increase in patients having duodenal ulcer and gastrinoma.

The prerequisite for developing a duodenal ulcer seems to be presence of an adequate amount of acid secretion. The occurrence of duodenal ulcer is rare in patients with a maximal acid output which is lesser than 11 to 14 mmol/hour.

Type I and type IV gastric ulcers in which excessive acid secretion is not found, acid may act like a cofactor, exacerbating ulcer damage already present and interfering with stomach`s heal abilities.

Type II or type III gastric ulcer patients have gastric acid hypersecretion and so they behave more like duodenal ulcers.

Duodenal Ulcer Pathophysiology

Duodenal ulcer is considered as a disease with numerous etiological factors. Hypersecretion of acid and pepsin along with either infection with H. pylori or NSAIDs intake is considered as an absolute requirement for duodenal ulcer development.

A number of secretory abnormalities are present in these patients , but the same abnormalities are not present in all patients . The common secretory abnormalities are

reduced bicarbonate secretion

increase in night time acid secretion

increase in duodenal acid load

increased gastrin sensitivity.

increase in mean parietal cell number

Gastric Ulcer Pathophysiology

Gastric ulcers usually occur near the incisuraangularis on the lesser curvature . About 65% of gastric ulcers are found in this region are called as type I ulcers. Type I ulcers are associated with a low or normal output of acid. They occur in the region of histologic transition zone of about 1.5 cms between that of antral and body gastric mucosa.

Type II gastric ulcers which occur in about 15% patients of gastic ulcer are present in the body of stomach in addition with presence of duodenal ulcer. These ulcers occur in association with excess acid secretion.

Type III gastric ulcers are called prepyloriculcers. They are found in around 25% patients with gastric ulcers. These ulcers are similar to duodenal ulcers as there is excess acid secretion.

Type IV gastric ulcers occur on lesser curvature region near gastroesophageal junction. These type IV gastric ulcers have a incidence of about 5%. These ulcers does not have increased secretion of acid.

Gastric ulcers usually develop after the age of 40 years. Their incidence peaks between the age of 55 and 65 years. Gastric ulcers occur more often in lower compared to higher social economic class .

Few conditions which predisposes to gastric ulcer formation are as follows

older age above 45

female sex

NSAIDs intake

Acid and pepsin secretory abnormalities

gastric stasis as a result of delayed gastric emptying

presence of duodenal gastric reflux of bile

presence of h.pylori infection.

Other conditions that may be a predisposing factor to gastric ulcer are smoking, chronic intake of alcohol , corticosteroid .

Rapid healing of these ulcers followantisecretory therapy, antacid therapy, or vagotomy.

CLINICAL FEATURES

Duodenal Ulcer

Duodenal ulcer patients may present in following ways

Abdominal Pain

This is the most common symptom in a duodenal ulcer patient. The pain is typically well localized occuring in the midepigastric region . Usually, it is a tolerable pain and most of the times relieved on food intake. The pain can sometimes be occurring episodically or seasonally during spring and winter, or it is relapsing at times of emotional distress. In case the pain is constantly present, it points towards deeper penetration of ulcer, and if there is pain referred to back it suggests pancreatic penetration by the ulcer.

Perforation

Around 5% of time, a duodenal ulcer will penetrate through the duodenum wall into the peritoneal cavity . This can produce chemical peritonitis. Perforation of an ulcer is frequently associated with fever,dehydration,tachycardia and ileus. Abdominal examination will reveal tenderness, rigidity, and rebound tenderness.

Bleeding

Bleeding from a peptic ulcer is the commonest cause of death in these patients . Duodenal ulcers on the posterior wall commonly bleeds . This is because of the thegastroduodenal artery lying directly posterior to duodenum. However , fortunately, many duodenal ulcers are superficially located or are found on parts of duodenum which are not near the gastroduodenal artery or the branches of the artery. As a result, minor bleeding is the most common presentation of most duodenal ulcers .

Obstruction

Duodenal inflammation due to ulcer can cause gastric outlet obstruction which results in a delay in gastric emptying, loss of appetite, and nausea associated vomiting. In case of patients with vomiting for prolonged periods, they will develop dehydration and can also lead to hypokalemichypochloremicmetabolic alkalosis due to loss of chloride, hydrogen, and potassium ions contained the gastric juice.

Chronic cases will have episodes of recurrent healing with repair and scarring. This will result in fibrosis, and hence narrowing of lumen

of duodenum . Marked loss of weight and malnutrition occur commonly in these chronically affected patients.

Gastric Ulcer

Gastric ulcer represent a challenging clinical situation because most of the time it may be impossible to differentiate between benign ulcer and carcinoma.

Similar to duodenal ulcers, gastric ulcers also can occur as recurring periods of quinescence and relapse. Gastric ulcers like duodenal ulcers cause pain but it is aggravated by food intake.

Bleeding, obstruction and perforation are the other presentations. Rarely , benign ulcers have caused spontaneous gastrocolic fistula.

DIAGNOSIS

Routine lab test includes a complete blood count, liver function test and a renal function testing with serum creatinine .

Serum gastrin assay is indicated in patient who are with ulcers which are not responding to medical treatment or in those requiring surgery.

A chest X-ray erect view may be done if perforation is suspected. The main method for diagnosis of peptic ulcer is fiberoptic endoscopy. Helicobacter pylori testing is mandatory for all patients who are suspected to have PUD.

H. pylori Testing

H. pyloritesting may be divided invasive and non-invasive tests . Non-invasive tests include

1. Serology
2. Carbon-labelled urea breath test.

Invasive tests require endoscopy and include

1. Rapid urease test
2. Histological testing
3. Culture.

Serology

Since *H. pylori* infection elicit a local and systemic immune response, serology is used for diagnosis of *Helicobacter pylori*. Serology is considered as the investigation of choice if endoscopic facilities are not available or not indicated It has sensitivity and specificity of about 90% . The limitations are because antibody titers remain high for about a year , so serology is not useful in determining eradication after treatment.

Urea Breath Test

A non-invasive test which is useful in diagnosis of *Helicobacter pylori*is carbon-labelled urea breath test. The basis of this depends on ability of helicobacter pylori to produce urease enzyme that hydrolyzes urea. It has a sensitivity and specificity of about 96%.

In this test the patient ingests C14 or C13 isotope labelled urea . After ingestion if H.pylori infection is present the carbon isotope labelled urea gets converted into ammonia and labelled bicarbonate . This labelled bicarbonate gets excreted through breath as labelled CO2, which can be detected. This is the test of choice for documenting eradication.

Rapid Urease Assay

This is the test of choice for diagnosing Helicobacterpyloriinfection when endoscopy is used. This is also a test based on *H. pylori* `s ability to hydrolyze urea. ureasecatalyzes urea to ammonia and bicarbonate, leading to creation of an environment alkaline in nature which can be identified by means of a pH indicator. Endoscopically obtained gastric mucosal biopsy is kept in a medium that contains urea and pH indicator and colour change observed. Sensitivity and specificity are around 95% and 97% respectively.

Histology

Biopsy samples are obtained by means of endoscopy from gastric mucosa which are histologically examined for H*elicobacter pylori. It* is identified by appearance with routinely used eosin and hematoxylin stains or by means of stains like silver or Giemsa . Sensitivity is 94% and specificity 98%. This test is used in assessing the extent of damage to gastric mucosa and also for confirmation of presence of H.pylori.

Culture

Culture of gastric mucosal biopsy samples obtained by performing an endoscopy can be used for diagnosis of*H. pylori*. The sensitivity is about 82% , specificity is 100%. Culture requires sophisticated laboratory, which is not available widely, and it requires about 4 to 6 days for diagnosis to be made. The main advantage is that it provides opportunity for performing antibiotic sensitivity testing if the need arise.

Upper Gastrointestinal Radiography

Peptic ulcer diagnosis using upper GI radiographs require findings like -demonstrating barium within ulcer crater, that may be oval or round with surrounding edema. Radiography also demonstrates the site and depth of ulcer and also the deformation resulting from chronic fibrosis if present.

Double-contrast techniques are used routinely which can detect 85% to 90% of ulcers as compared to single contrast techniques in which detection rate of ulcers is only about 50% . Ulcer size may have some predictive value in which larger lesions have higher possibility of malignancy as compared to smaller ones. An ulcer that has irregular filling defects is more in favour of a malignant ulcer.

Fibroptic Endoscopy

The most reliable method for diagnosis of a peptic ulcer is by means of endoscopy.

Benign ulcers appear to have a smooth, rounded , regular margins associated with base that is flat.

Malignant ulcers are commonly associated with mass protruding into the lumen or may have folds around the ulcer which stop short of the margin of ulcer. Biopsy is required from the ulcers to rule out malignancy .

Endoscopy also provides tissue for testing *Helicobacter pylori* infection and can be used therapeutically in patients with GI bleed .

TREATMENT

Medical treatment:

Patients with symptoms related to ulcer are usually treated with H2 antagonists or proton pump inhibitors(PPI) before upper GI endoscopy is done. If symptoms persists an endoscopy is indicated.

Patients on NSAIDs can be given prostaglandin analogues like Misoprostol for prevention of peptic ulcers.

If H. pylori infection is identified, its effective treatment is by a combination of 2 antibiotics (e.g.Amoxicillin,clarithromycin, Metronidazole,tetracycline) and 1 PPI (omeprazole, rabeprazole, lanzoprazole) .

In treatment-resistant patients, three antibiotics (e.g. amoxicillin + metronidazole +clarithromycin) can be used along with a PPI and sometimes together with bismuth compound. H. pylori treatment usually will lead to clearance of infection, leading to healing of ulcers.

If H. pylori is not identified, long-term PPIs are used with search for other causes of ulcer.

With widespread use of PPI's from the 1991 and identification of h.pylori`s role in PUD , surgical procedures in case of uncomplicated peptic ulcers have became obsolete now

Surgeries for Peptic Ulcer :

The indication for surgery in peptic ulcer disease are

1.Intractability

2. Perforation

3. Bleeding

4.obstruction.

Elective surgeries have become rare as medical therapy has becomes more effective.

TruncalVagotomy

Truncalvagotomy is done by dividing the anterior and posterior vagal nerves above the gastroesophageal junction. It is the most commonly done surgery for duodenal ulcer. Drainage procedure is usually done in combination with a truncalvagotomy which can be the

classical Heineke-Mikuliczpyloroplasty or Finney pyloroplasty or Jaboulaygastroduodenostomy .

Highly Selective Vagotomy (Parietal Cell Vagotomy)

The highly selective vagotomy is otherwise called *proximal cell vagotomy*or*parietal cell vagotomy*. In highly selective vagotomy only the vagal nerve fibres which supply the acid-producing part of stomach in the corpus and fundus are divided. As this procedure preserves antral innervation, drainage procedures are not needed. As a result, postoperative complications are also less.

Recurrence rates of about 10% to 14% are reported for this procedure which are higher compared with truncalvagotomy and pyloroplasty (recurrence rate- 5 to 8%). But, truncalvagotomy with drainage procedures are commonly related with dumping syndrome and diarrhea. Because of these factors, recurrence rate associated with parietal cell vagotomy is considered acceptable due to the fact that recurrences are responsive to medical therapy with PPI.

TruncalVagotomy and Antrectomy

Antrectomy is indicated in patients with distal gastric ulcer. Antrectomy requires reconstruction which can be done either by means of a gastro- duodenal anastomosis (Billroth I procedure) or a gastrojejunal anastomosis (Billroth II procedure) .

When it is combined with truncalvagotomy, it is superior to both truncalvagotomy with drainage procedure or highly selective vagotomy at decreasing secretion of acid and also ulcer recurrence rates . The ulcer recurrence rates following truncalvagotomy and antrectomy is about 0.5% to 1%. But, low ulcer recurrence rates has to be balanced against complications which is about 20%.

Summary of surgical treatment recommended for intractable peptic ulcer disease.

Duodenal ulcer: Highly selective or parietal cell vagotomy.

Gastric ulcer

Type I : Distal gastrectomy or anterectomy with billroth I anastomosis

Type II or III : Distal gastrectomy or anterectomy with billroth I anastomosis along with truncalvagotomy

Type IV : The surgical treatment is based on ulcer size, distance from gastro-esophageal junction and also amount of inflammation surrounding the ulcer. The most commonly performed procedure is a gastrectomy that include the ulcer followed by Roux-en-Y esophogogastrojejunostomy to restore continuity

PERFORATED PEPTIC ULCER

EPIDEMIOLOGY:

Perforation of peptic ulcer occur in 2-10% of patients with PUD and this account for about 70% of deaths occurring due to PUD. Perforation may sometime be the first clinical presentation of PUD.

In male, incidence of ulcer perforation increased till about 1950 and declined thereafter . Contrasting to this, in women the incidence was low and stable till about 1950, from then it has slowly increased . Age among ulcer perforation patients increased, with incidence declining among young people and increasing among elderly . The male to female ratio has decreased from 10:1 to 1.4 : 1 and the median age has increased from 30 to above 60. Life time risk of perforation in peptic ulcer patients who do not receive any treatment is considered to be about 10%.

The incidence of peptic ulcer perforation is 7-10 cases/ 100.000 per year. The perforation site may involve the anterior wall of duodenum (60%),antrum (25%) and lesser-curvature gastric ulcers(15%).

The need for surgery in case of perforation has remained stable and the mortality have not decreased even after the introduction of H2 receptor antagonists. Peptic ulcer perforation still causes about 20,000-30,000 deaths annually in Europe.

PATHOGENESIS

The pathogenesis of peptic ulcer disease is best considered as representation of because of imbalance occuring between defensive (mucous-bicarbonate layer, prostaglandins) and aggressive factors (acid, pepsin, ethanol, smoking etc.) .

In more recent years Helicobacter pylori(H.pylori) and NSAIDs have been identified as two main causes of peptic ulcer. The crack cocaine usage has also led to increase in Perforation rates, but it has a different mechanism since PPU secondary to the use of crack cocaine is because of ischemia of gastric mucosa and during treatment , these perforations do not warrant an acid reducing surgery .

Three clinical phases have been identified in the process of PPU.

Phase 1: Chemical peritonitis :

Perforation leads to chemical peritonitis due to acid resulting in intense,diffuse abdominal pain.

Phase 2: Intermediate stage:

Occurs after a period of 7-10 hrs, patients obtain some amount of spontaneous relief of pain. This is because of dilution of irritatinggastic contents by peritoneal exudative secretion.

Phase 3: Intra-abdominal infection:

after about 11-24 hrs infection supervenes and patients condition worsens.

ETIOLOGY

The main risk factors for development of perforation of peptic ulcer disease are

H.pylori

NSAID usage

Cigarette smoking

Cocaine usage

CLINICAL PRESENTATION

Perforation is characterised by severe and sudden epigastric abdominal pain which progresses to involve the entire abdomen. This pain rapidly reaches peak intensity and remains constant till peritoneal reactionary stage, after which it worsens.

Radiation to scapula and shoulder on the right side indicate right subcostal collection of contents.

Valentino's syndrome

This is pain presenting in right lower quadrant of abdomen mimicking acute appendicitis but actually caused by duodenal ulcer perforation with gastric contents collecting in right paracolic gutter.

It is named after the famous Italian actor Rudolph Valentino who presented with perforated peptic ulcer and right lower quadrant pain. He died subsequently following surgery.

The degree of peritoneal irritation is influenced by

Perforation size

Amount of gastric and bacterial contents contaminating the peritoneal cavity

Time between perforation and presentation

Physical findings:

Low grade fever, diffuse abdominal tenderness , guarding and rigidy of abdominal wall, diminished movement of the abdomen, diminished or absent bowel sounds, obliteration of liver dullness.

The patient may also present with features of early shock like tachycardia, hypotension and decrease in urine output.

DIFFERENTIAL DIAGNOSIS

Acute pancreatitis

Acute cholecystitis

Dissecting aortic aneurysm

Acute myocardial infarction

Acute appendicitis

Acute diverticulitis

RISK STRATIFICATION

There are a number of scoring systems available for predicting the outcome due to perforated peptic ulcer. Some of them are APACHE II, Mannheimer peritonitis index, PULP scoring, ASA scoring. The most simple of the scoring system is Boey scoring symptoms which includes –

Delay in presentation (>24 hours)

Pre operative shock (BP < 90 mmhg)

Associated serious medical illness

With these scoring symptoms patients can stratified and prediction mortality and morbidity can be done.

INVESTIGATIONS

Moderate leulocytosis on complete blood count

Chest x-ray abdomen erect or lateral abdominal decubitus x-ray:

Free air under diaghragm suggesting pneumoperitoneum is found about 70 to 80% of patients

Upper GI study:

gastrograffin , a water soluble contrast ,can show extravasation of contrast if the perforation has not sealed spontaneously. This is a useful study if non-operative management is contemplated.

CT scan with contrast:

can show extravsation of contrast. Can help in differential diagnosis when in doubt

Upper GI endoscopy:

Currently not recommended because it has the potential to disrupt spontaneous sealing of perforation.

TREATMENT

As soon as a diagnosis of perforated peptic ulcer is made, resuscitation should be started immediately with large volume of crystalloids, nasogastric suction to keep the stomach empty; and broadspectrum antibiotics administration.

When perforated peptic ulcer is diagnosed, few different therapeutic options available, are to be taken into consideration. At first it should be evaluated that, is the patient suitable candidate for surgery or instead conservative treatment can be considered .

If surgery is planned, is it sufficient to perform simple closure with or without omentoplastyor a definitive surgery for ulcer is to be considered and also if definitive surgery is being done, which specifc operation is it to be done? Final consideration to be taken into account is, can the surgery be done laparoscopically or laparotomy a safer option?

Non operative management or Conservative treatment :

This is known as the Taylor method and it consists of nasogastric aspiration,intravenousfluids,antibiotics and in recent years include H.pyloritriple therapy . In the year 1946, Taylor presented his study on a series of patients who had successfully outcome following conservative treatment of PPU, based upon the theory, that effective gastric decompression and continuous drainage enhances self healing.

Since then many reports on this topic have been published, with variable success rates. But still, debate continues whether PPU needs to be generally operated on or not.

Indications of non operative treatment:

Patients < 70 years, not suitable for surgical repair due to associatedCo- morbidity, with documented contrast study that shows completely sealed perforation.

Contra-indications for non operative treatment:

patients in shock

Time point between perforation and start treatment of treatment > 24 hours

Patients > 70 years

Advantages :

Avoidance of surgery with its associated morbidity like that of intraabdominal adhesions caused by surgery which later on makes elective surgery for PUD difficult and also anaesthesia related complications

Disadvantages:

Higher rate of mortality if conservative treatment is to fail.

Lack of benefit of laparotomy or laparoscopy as diagnostic tool in case patient was misdiagnosed.

4% chance of intra-abdominal abscess and 3% chance of reperforation.

Finally, one should always bear in their mind that perforated gastric ulcer can be a presentation of gastric cancer, so if one has chosen conservative treatment, after a few weeks endoscopy is mandatory.

Simple suture Open repair technique:

During all surgical procedures for PPU prophylactic antibiotics must be given at induction of anesthesia. In convention, an uppermidlinelapatotomy incision is made and abdomen opened. Then search for site of perforation is done , identification of perforation site is always not easy, because sometimes a perforation might have occurred on the posterior aspect of stomach, only to be detected if opening of lesser sac is done. Also double perforations might occur. If perforation is by a gastric ulcer a biopsy is mandatory to exclude gastric cancer.

Simple closure of perforation can be done in following ways

1. Primary closure by interrupted Sutures

2.Primary closure by interrupted sutures covered with pedicledomentoplasty

Cellan-Jones repair:

Plugging the perforation by pedicledomentoplasty

Graham patch:

Plugging the perforation by free omental plug

Treatment of choice initailly was, excision of friable edges and the applying of purse string sutures to close the defect and an omental graft on top of it . Anproblem encountered with this is narrowing of duodenum. To avoid this, omentoplasty without primary closing of defect is now recommended as the treatment of choice. The omental graft promotes healing by the stimulus for formation of fibrin.

Thorough peritoneal toilet is performed then. One or two drains are placed and then wound closed.

Definitive surgery:

Indications for performing elective surgery are still not clearly defined .only about 0.3% of surgeons routinely perform a vagotomy for duodenal ulcer perforation.

Reasons for decline in performing definitive ulcer surgery are because of lower recurrence as a result of H.pylori eradication and elimination of NSAID usage post-operatively. Also that, patients operated for PPU are older having higher surgical risk make them less suitable for definitive ulcer surgery.

Patients in whom definitive ulcer surgery is to be considered are those with PPU who are H.pylori negative, or in those with recurrent ulcers despite treatment. In this patients a parietal cell vagotomy is recommended.

Laparoscopy:

laparoscopic closure of perforated peptic ulcer hasbeen practised since 90`s.

Advantages:

laparoscopic procedure serve as a minimal invasive diagnostic tool.

Laparoscopic repair are associated with less postoperative pain and decreased use of analgesics and a shorter duration of hospital stay.

There is also a reduction in wound infections, development of burst abdomen and also incisional hernia due to shorter scars.

lower incidence of postoperative ileus and also chest infections .

Drawbacks:

Prolonged operating time

Greater incidence of re-operations as compared to open method due to leakage at repair site because of difficulty in laparascopy suturing techniques.

Higher incidence of intra-abdominal collection due to inadequate lavage.

Recently suture less techniques are being tried, in which fibrin glue or a gelatine sponge has been placed and glued into the ulcer . The drawback of this is that only for small perforations this can be used. To overcome this, a biodegradable patch, which can be cut into any desirable shape and size, has been tested in rats, and has shown good results.

Post operative complications:

Complication	**Incidence**
Pneumonia	3.7-30%
Wound infection	11-17%
Urinary tract infection	1.5-15%
Suture leak	3-16%
Abscess formation	0-8%
Heart problems (myocardial infarction, heart failure)	5.2%
Ileus	2-5%
Fistula	0.6-4%
Wound dehiscence	2.6-6%
Biliary leak	3.9%
Bleeding	0.7%
Re-operation	3-9%
Sepsis	2.6%
Stroke	4%
Death	6-11%

Postoperative management :

All patients should receive nasogastric tube aspiration for at least 48 hrs .oral feeding can be started on 3rd day according to the recent Cochrane review.

Preoperative and postoperative intravenous administration of antibiotics have proven to decrease the overall infection rate .

Treatment of H. pylori infection with triple therapy consisting of a PPI, amoxicillin and clarithromycin for a period 14 days is recommended. Upper GI endoscopy is to be performed after eight weeks in order to asses healing of ulcers and also to check for H.pylori eradication.

CONCLUSION

Surgery for perforated peptic ulcer is still a subject with lot of debate despite nearly an era of published data. Reviewing different policies , for instance , indication for conservative treatment, the need for omentoplasty , performance of the procedure laparoscopically and need for definitive ulcer surgery, might help in establishing consensus.

MATERIALS AND METHODS

A prospective observational study was conducted in our department for a period of 1 year from November 2012 to November 2013.

A total of 50 patient were included in this study.

Inclusion criteria*:*

All patients with perforated peptic ulcer managed surgically.

Exclusion criteria*:*

Patients with perforated peptic ulcer disease managed conservatively.

All patients in whom a diagnosis of perforated peptic ulcer is made are included in this study after getting informed consent from the patient. .

In all these patients Boey score was calculated prior to surgery. Boey`s score includes

presence of pre-operative shock(systolic BP < 90mm hg),

time of perforation prior to surgery >24 hours,

concomitant severe medical illness.

A score ranging from 0 to 3 was given to the patients depending on the above criteria .

These patients were managed surgically by upper midline laparotomy and omental patch closure of the perforation.

All these patients received pre and post operativeantibiotics .

Post operatively observed mortality and morbidity of the patients were co-related with boey score and conclusions were drawn using statistical methods.

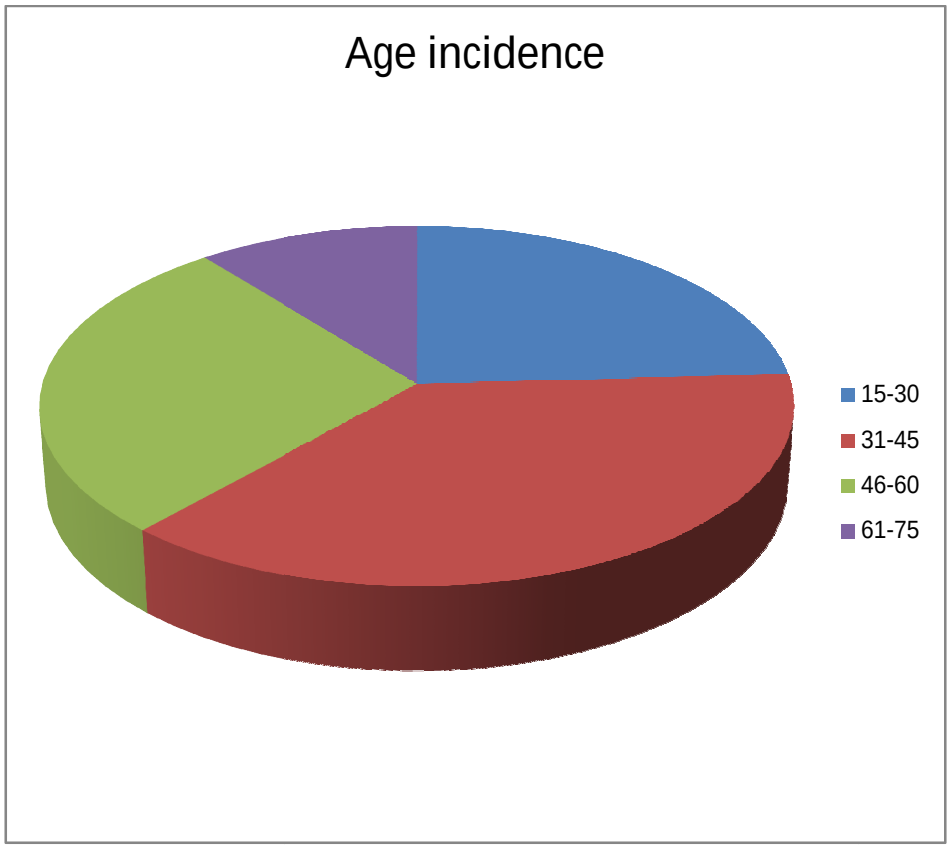
OBSERVATION AND RESULTS

Age incidence:

A total of 50 patients were included in this study out of which the lowest age was 16 and highest was 70.

The age group distribution is

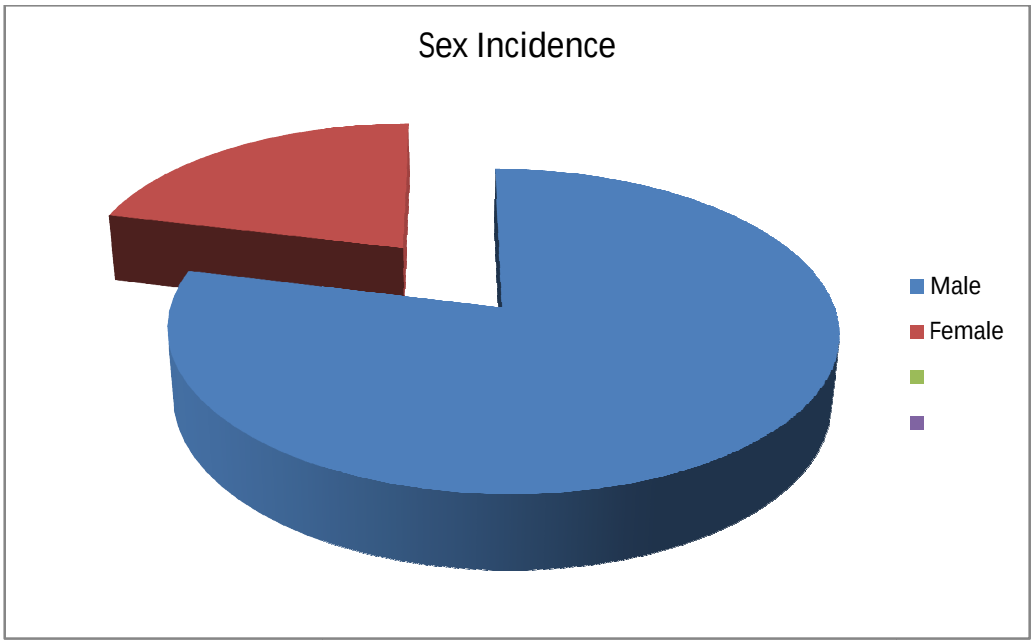
Age interval	No. of patients
15-30	**12**
31-45	**17**
46-60	**14**
61-75	**7**



This above pie chart shows the different age distribution of patients ranging from age of 15 – 75.

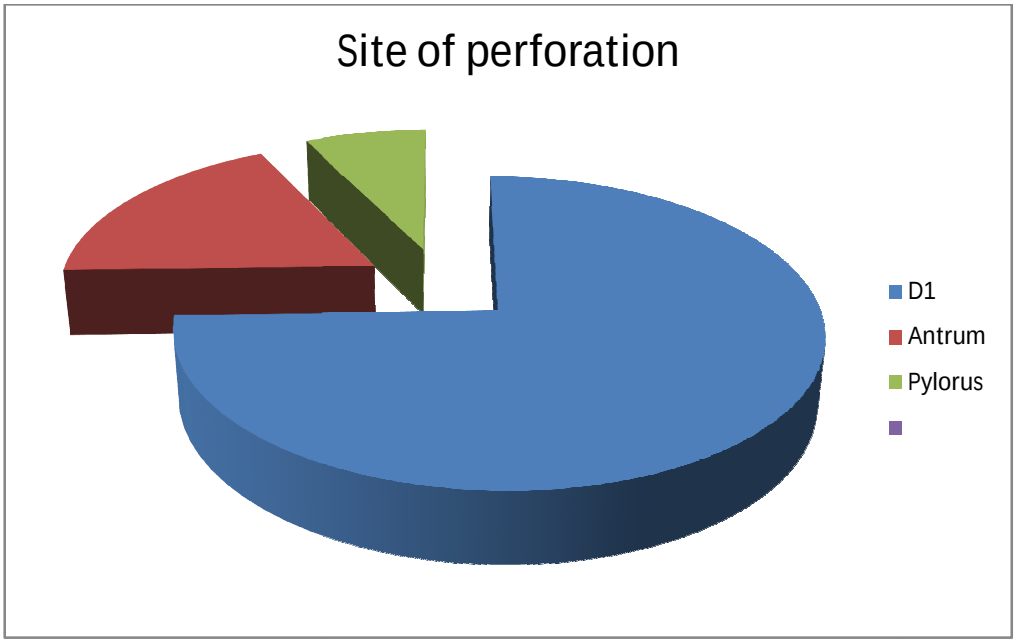
Sex Incidence:

Male	Female
44	6



Site of perforation:

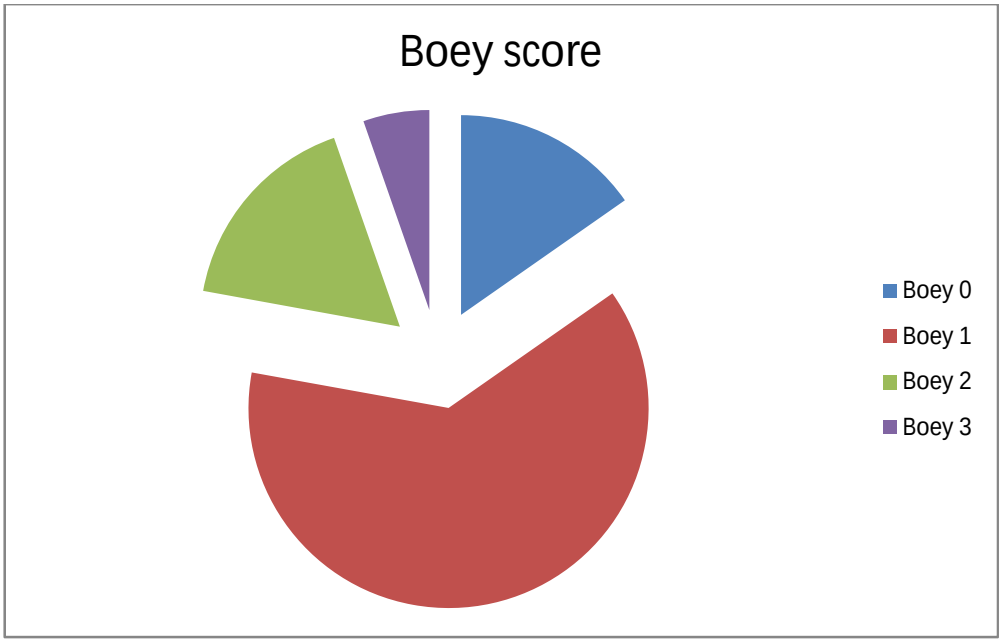
Site of perforation	No.of patients
First part of duodenum	40
antrum	8
pylorus	2



Boey score:

The representation of boey score of the 50 patients in this study is as follows

Boey score	No. of patients
0	8
1	29
2	11
3	2

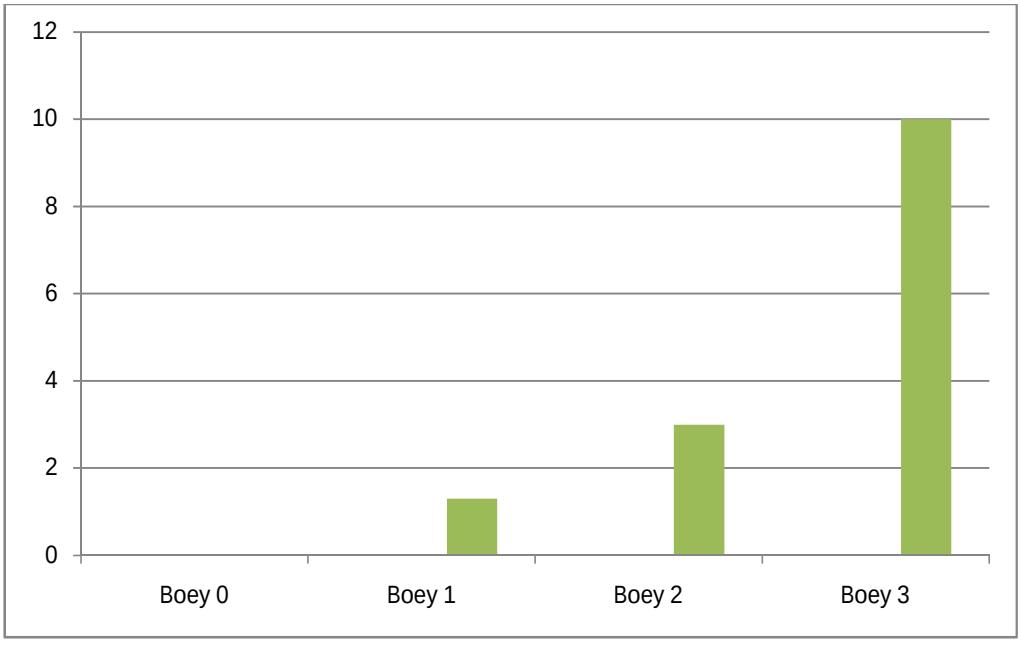


Mortality rate and Boey score comparison:

9 patients out of 50 died post – operatively.

Boey score	No. of patients who expired	Mortality Rate
0	0	
1	4	13%
2	3	27%
3	2	100%

Boey score and mortality rate depicted in this following chart



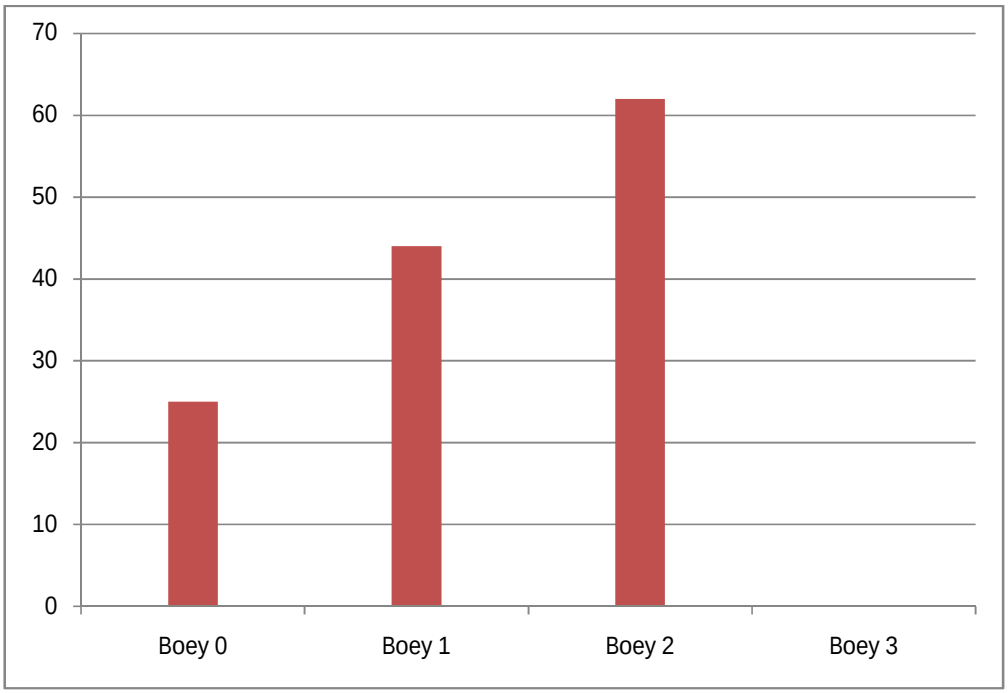
Morbidity:

The following is the list of complications and the incidence of each complications which the patients developed.

Morbidity	No. of patients with morbidity	Incidence
Wound infection	15	36.5%
Respiratory tract infection	11	26.8%
Abscess formation (pelvic,subdiaphragmatic)	3	7%
Wound dehiscence	2	4%
Suture leak (re-perforation)	1	1.7%
Urinary tract infection	3	7%

Comparison between Boey score and Morbidity rate:

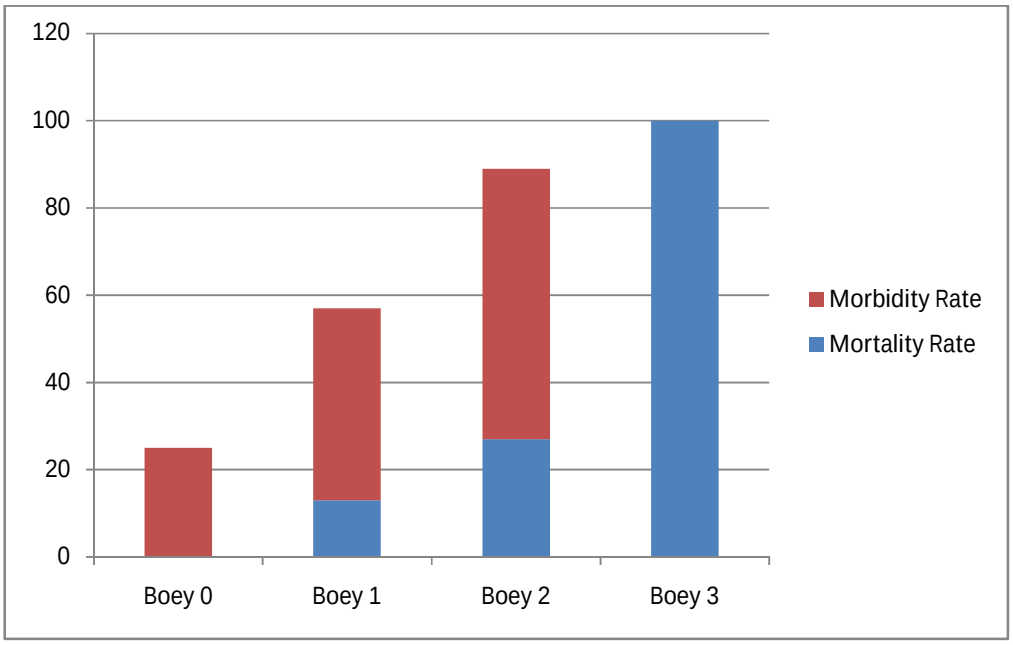
Boey score	No.of patients with morbidity	Morbidity Rate
0	2	25%
1	11	44%
2	5	62%
3	-	



Boey score related morbidity is depicted in the above chart

Final scoring comparing Boey score and Mortality and Morbidity rate:

Boey Score	Mortality rate	Morbidity rate
0	-	25%
1	13%	44%
2	27%	62%
3	100%	-



DISCUSSION

In the original study done in the year 1987 by Boey the results are as follows

Boey score	Morbidity rate	Moratlity rate
0	17.4%	1.5%
1	30.1%	14.4%
2	42.1%	32.1%
3		100%

In the most recent years this scoring has been used for predicting post-operative mortality and results of some this studies are as below:

Author	year	Mortality rate Boey 0	Mortality rate Boey 1	Mortality rate Boey 2	Mortality rate Boey 3
Lee FYJ	2001	0	14%	32%	100%
Arici C	2006	1.5%	12%	33%	64%
Lohsiriwat V	2008	0	8%	31%	42%

In our hospital the study was done over a period of 12 months and the results of mortality and morbidity rate obtained in our setup have been depicted already.

Comparing this study with the original study done by Boey the resuts are almost similar except for slightly increased morbidity rates observed in patients with scores 1 and 2.

When compared with the most recent studies the difference is mainly in the mortality rates of patients with score of 3 where the mortality rates were around 45-60% as compared to 100% in our study.

This large disparity is due to the fact that only less number of patients wereincluded and only 2 patients had a score of 3. The other reason being both of these patients were older than 65 years and there is evidence that risk of mortality increases in patients older than 60 years of age.

CONCLUSION

Boey score being a very simple score and easy to calculate, can be used for risk stratification of patient with perforated peptic ulcer, so that patients with higher risk as decided by higher scores can receive greater intensive care.

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PROFORMA

ROLE OF BOEY SCORE IN PREDICTING MORTALITY AND MORBIDITY IN PERFORATED PEPTIC ULCER

INVESTIGATOR: DR.DARWIN BRITTO.D PGY3 MS GEN SUR

GUIDE: PROF.DR.BALAMURUGAN`s CHIEF S7 UNIT

Name: Age/ Sex: IP NO:

Address:
Contact no:

D.O.A: D.O.S: D.O.D:

CHIEF COMPLAINTS AND RELEVANT HISTORY:

VITAL SIGNS:

SYSTEMIC EXAMINATIONS:

Cvs/rs:

PA:

Boey score:

Time since perforation:

Presence of pre-operative shock:

Concomitant severe medical illness:

INVESTIGATION:

HB -

PCV-

TC –

DC –

ESR –

RBS –

BLOOD UREA –

SERUM CREATININE –

SERUM ELECTOLYTES –

X-RAY CHEST

X-RAY ABDOMEN –

ECG –

INTRA-OPERATIVE FINDINGS:

POST OPERATIVE FOLLOW UP:

ROLE OF BOEY SCORE IN PREDICTING MORTALITY AND MORBIDITY IN PERFORATED PEPTIC ULCER

INVESTIGATOR: DR.DARWIN BRITTO.D MS GEN SUR

GUIDE: PROF.DR.BALAMURUGAN`s CHIEF S7 UNIT

PATIENT INFORMATION MODULE

You are being invited to be a subject in this study.

Before you participate in this study, I am giving you the following details about this trial, which includes the aims, methodology, intervention, possible side effects, if any and outcomes.

All patients with a diagnosis of perforated peptic ulcer will be included in this study.A detailed clinical history will be taken following a standardized proforma. A detailed clinical examination will be made and relevant investigations will be done .Boey score will be calculated pre-operatively. This score includes presence of pre-operative shock(systolic bp<90), time of perforation >24 hours, concomitant severe medical illness. Patients will be subjected to surgery and followed up post-operatively.

The results arising from this study will be analyzed and used for academic purposes. You will be given clear instructions at every step and you are free to ask/ clarify any doubts. Your identity will remain confidential. You are free to withdraw from this trial at any point of time, without any prior notice &/ or without any medical or legal implications.

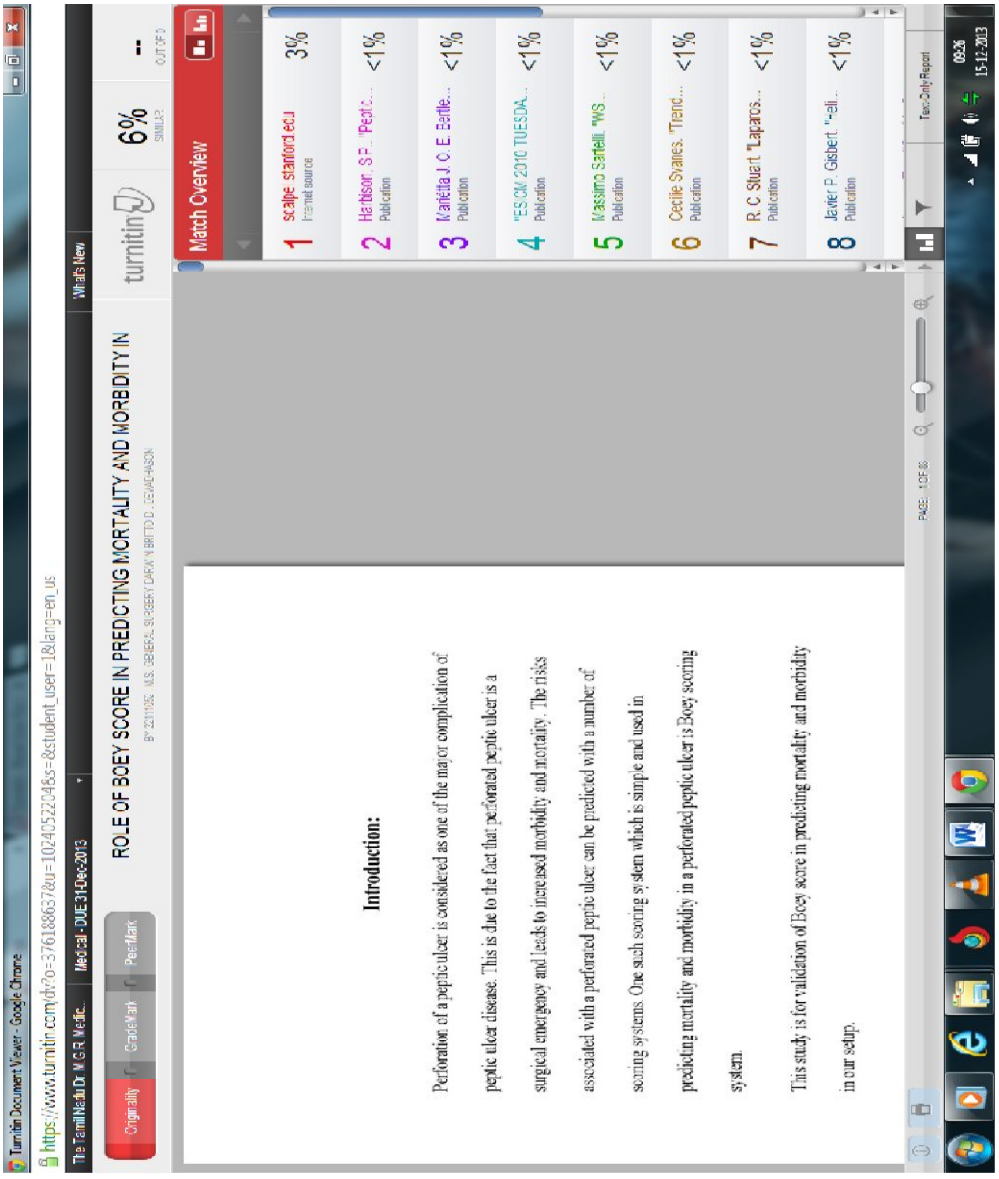
I request you to volunteer for this study.

Thanking you

Investigator's Sign Patient's Sign

(DR.D.DARWIN BRITTO) (NAME:)

TURN IT IN SCREENSHOT :



s.no	Name	age/sex	IP.No	Boey score	Mortality	wound inf.	res. Inf.	abscess forma.	wound dehiscence	suture leak	UTI
1	Shanmugam	31/M	54062	0	-	yes	yes	no	no	no	no
2	Elumalai	55/M	55321	2	yes						
3	Mariyaraj	40/M	59420	1	-	no	no	no	no	no	no
4	Venkatalakshmi	50/F	1	1	-	no	yes	no	no	no	no
5	Muruganantham	28/M	716	1	-	yes	no	no	no	no	yes
6	Elumalai	40/M	903	0	-	no	no	no	no	no	no
7	Krishnan	50/M	1107	1	-	no	no	no	no	no	no
8	Siva	20/M	1138	1	-	no	no	no	no	no	no
9	Ramesh	45/M	2579	2	-	yes	yes	yes	no	no	no
10	Sekar	42/M	2644	1	yes						
11	Kuselan	50/M	3263	3	yes						
12	Kumar	54/M	3714	0	-	no	no	no	no	no	no
13	Sanjeevi	38/M	4349	1	-	no	yes	no	no	no	no
14	Dhanakodi	60/F	4352	1	-	no	yes	no	no	no	no
15	Rajendran	30/M	7113	1	-	no	no	no	no	no	no
16	Abdul Hakeem	55/M	7950	0	-	no	no	no	no	no	no
17	Sasikumar	27/M	8364	1	-	no	no	no	no	no	no
18	Durairaj	65/M	8538	2	-	yes	yes	no	no	no	no
19	Mythili	36/F	8883	1	-	yes	no	no	yes	no	no
20	Subbaih	47/M	9312	1	yes						
21	Mohan Raj	16/M	10204	1	-	no	no	no	no	no	no
22	Prakash	45/M	12531	0	-	yes	yes	no	no	no	no
23	George	40/M	14221	2	yes						
24	Haridoss	62/M	14279	1	yes						
25	Bhagavan singh	42/M	14602	1	-	yes	yes	no	no	no	no
26	Mariyammal	65/F	15149	3	yes						
27	Ragavendran	25/M	16049	1	-	no	no	no	no	no	no
28	Pandiyan	50/M	16763	1	-	no	no	no	no	no	no
29	Murali	27/M	18471	0	-	no	no	no	no	no	no
30	Saleem	37/M	20698	2	-	yes	yes	no	no	no	no
31	Chandran	28/M	22195	1	-	no	no	no	no	no	no
32	Aalaiyammal	53/F	23544	2	-	yes	no	yes	no	no	no
33	Sivalingam	65/M	24657	1	-	no	no	no	no	no	no
34	Sudha	28/F	26910	2	-	yes	no	no	no	no	yes
35	Chinnaiya	44/M	28884	1	-	yes	yes	no	no	no	no

36 Murugan	50/M	30555	1	yes						
37 Abdul kathias	56/M	31963	2	-	no	no	no	no	no	no
38 Velayudham	43/M	34308	1	-	yes	no	no	no	yes	no
39 Elumalai	27/M	35306	0	-	no	no	no	no	no	no
40 Manimaran	38/M	36552	1	-	no	no	no	no	no	no
41 Selvam	35/M	37897	2	-	no	no	no	no	no	no
42 Hari	33/M	39148	1	-	yes	no	yes	no	no	no
43 Srinivasan	22/M	40221	0	-	no	no	no	no	no	no
44 Prasanth	21/M	42031	1	-	no	no	no	no	no	no
45 Ranjith Kumar	20/M	42522	1	-	no	no	no	no	no	no
46 Raavanan	55/M	44089	2	-	no	no	no	no	no	no
47 Subramani	55/M	44304	1	-	yes	no	no	yes	no	yes
48 Manoharan	30/M	46333	1	-	no	no	no	no	no	no
49 Doss	70/M	46893	2	yes						
50 Maniram	35/M	47404	1	-	yes	yes	no	no	no	no

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Role of Boey score in predicting mortality and morbidity In perforated pepticulcer

Principal Investigator : Dr.D.Darwin Britto

Designation : PG in MS (General Surgery)

Department : Department of General Surgery
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.04.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

27/9/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI